(FILE 'HOME' ENTERED AT 10:03:35 ON 08 MAR 2004)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 10:03:51 ON 08 MAR 2004

L1 1967 S HCC (P) (TUMOR SUPPRESSOR OR P53 OR P34 OR P51 ORRB OR MPL)
L2 824 S HCC (5A) (TUMOR SUPPRESSOR OR P53 OR P34 OR P51 ORRB OR MPL)
L3 854 S HCC (5A) (TUMOR SUPPRESSOR OR P53 OR P34 OR P51 OR RB OR MPL)
L4 283 S L3 AND PY<1997
L5 158 S HCC (5A) (TUMOR SUPPRESSOR OR P34 OR P51 OR RB OR MPL)
L6 40 S L5 AND PY<1997
L7 19 DUP REM L6 (21 DUPLICATES REMOVED)

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L7 ANSWER 12 OF 19 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 94049586 EMBASE
DOCUMENT NUMBER: 1994049586
TITLE: Alterations of tumor suppressor genes and allelic losses in human hepatocellular carcinomas in China.
AUTHOR: Fujimoto Y.; Hampton L.L.; Wirth P.J.; Wang N.J.; Xie J.P.; Thorgeirsson S.S.
CORPORATE SOURCE: Lab. of Experimental Carcinogenesis, National Cancer Institute, Building 37,Bethesda, MD 20892, United States
SOURCE: Cancer Research, (1994) 54/1 (281-285).
ISSN: 0008-5472 CODEN: CNREA8
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Aflatoxin B1 has been suggested as a causative agent for a G to T mutation at codon 249 in the p53 gene in human hepatocellular carcinomas (HCC) from southern Africa and Qidong in China. The objective of the present work was to test the hypothesis that exposure to aflatoxin B1 either alone or coincident with other environmental carcinogens might be associated with allelic losses occurring during development of human hepatocarcinogenesis in China. The HCCs were obtained from two different areas in China: Qidong, where exposure to hepatitis B virus (HBV) and aflatoxin B1 is high; and Beijing, where exposure to HBV is high but that of aflatoxin B1 is low. We analyzed the tumors for mutations in the p53 gene and loss of heterozygosity for the p53, Rb, and APC genes and at marker loci on chromosomes 4, 13, and 16. Frequencies of mutation, loss, and aberration (mutation and loss) of the p53 gene in 25 HCCs from Qidong were 60, 58, and 80%, respectively. The frequencies in 9 HCCs from Beijing were 56, 57, and 78%. However, the frequency of a G to T transversion at codon 249 in HCCs from Qidong and Beijing were 52 and 0%, respectively. These data indicate that mutation and/or loss of heterozygosity in the p53 gene, independent of the 249 mutation, play a critical role in the development of hepatitis B virus- associated **HCCs** in China. Loss of the **Rb** and APC genes was observed in 44 and 7% of HCCs from Qidong, respectively. Allelic losses on chromosome 4 and especially on chromosome 16 were frequent in HCCs from Qidong but were not observed in HCCs from Beijing, while loss of heterozygosity on chromosome 13 occurred at similar frequency in both Qidong and Beijing. These results show a distinct difference in the pattern of allelic losses between HCCs in Qidong and Beijing and suggest that aflatoxin B1 and/or other environmental carcinogens may contribute to this difference.

L7 ANSWER 19 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1991:378518 BIOSIS
DOCUMENT NUMBER: PREV199141050908; BR41:50908
TITLE: DIFFERENTIAL EXPRESSION OF ONCOGENES AND **TUMOR SUPPRESSOR** GENE IN HUMAN HEPATOCELLULAR CARCINOMA **HCC** AND HEPATOBLASTOMA HB CELL LINES.
AUTHOR(S): FARSHID M [Reprint author]; TABOR E
CORPORATE SOURCE: NATIONAL INST HEALTH, BETHESDA, MD 20892, USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (1991) Vol. 32, pp. 278.
Meeting Info.: PROCEEDINGS OF THE 82ND ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR CANCER RESEARCH, HOUSTON, TEXAS, USA, MAY 15-18, 1991. PROC AM ASSOC CANCER RES ANNU MEET.
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 17 Aug 1991
Last Updated on STN: 17 Aug 1991

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L7 ANSWER 18 OF 19 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1991:200917 CAPLUS
DOCUMENT NUMBER: 114:200917
TITLE: Selective G to T mutations of p53 gene in hepatocellular carcinoma from southern Africa
AUTHOR(S): Bressac, Brigitte; Kew, Michael; Wands, Jack; Ozturk, Mehmet
CORPORATE SOURCE: Cancer Cent., Massachusetts Gen. Hosp., Charlestown, MA, 02129, USA
SOURCE: Nature (London, United Kingdom) (**1991**), 350(6317), 429-31
CODEN: NATUAS; ISSN: 0028-0836
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Hepatocellular carcinoma (HCC) is a prevalent cancer in sub-Saharran Africa and eastern Asia. Hepatitis B virus and aflatoxins are risk factors for HCC, but the mol. mechanism of human hepatocellular carcinogenesis is largely unknown. Abnormalities in the structure and expression of the **tumor-suppressor** gene p53 are frequent in **HCC** cell lines, and allelic losses from chromosome 17p have been found in HCCs from China and Japan. Allelic deletions from chromosome 17p and mutations of the p53 gene found in 50% of primary HCCs from Southern Africa are reported. Four of five mutations detected were G → T substitutions, with clustering at codon 249. This mutation specificity could reflect exposure to a specific carcinogen, one candidate being aflatoxin B1, a food contaminant in Africa, which is both a mutagen that induces G to T substitution and a liver-specific carcinogen.